THE SYMPTOMATOLOGY OF LESIONS OF THE LENTICULAR ZONE WITH SOME DISCUSSION OF THE PATHOLOGY OF APHASIA.*

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INTRODUCTION.

The views on aphasia recently advanced by Marie have served, among other things, to concentrate the attention of physiologists and neurologists upon the functions of the lenticular nucleus, to which body Marie has given an unusual role in the production of motor aphasia. It is a noteworthy fact that, in spite of the neurological activity all over the scientific world during the last three or four decades, the functions of the basal ganglia still remain largely undecided. Many speculations have been advanced regarding the functions of the thalamus, caudatum and lenticula; and many facts, anatomical, physiological and clinicopathological, have been contributed regarding these organs; but the comparative certainty of our knowledge as to the functions of the cerebellum, most of the cortical fields and centers, and many of the encephalic tracts of projection and association, is lacking with regard to these ganglia. The reasons for this state of affairs are not hard to see. Owing to the deeply seated position of the basal ganglia, it is difficult to experiment on them without involving or injuring other parts; and the lesions which are observed by the clinicopathologist are rarely absolutely limited to any one of these organs. Microscopical investigations of degenerations of these ganglia and of their incoming and outgoing tracts have not led to as clear inferences as in other regions of the brain.

Our paper is based in the first place upon a study of eleven cases with necropsies, in ten of which the lesion involved the lenticular zone. In six of these cases the lenticula itself was involved. Other parts of the lenticular zone were variously implicated in the ten cases. It was possible, however, at least in some of the cases, in which regions other than the lenticula were involved, to arrive at an idea as to the part played by this body in the symptomatology presented by the patients.

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For several years the writers of the paper have been collecting cases with necropsies in which lesions of the basal ganglia have been present. Most of the specimens from these cases are to be found in the Laboratory of Neuropathology of the University of Pennsylvania. In not a few instances papers have been published in which references have been made to some of these cases, and some use will be made of these published but personal data. In going over the specimens of cases, clinical and pathological studies of which have not yet been made, the writers found that they had in their possession more than twice as many specimens of cases as have been used in this paper, but owing to the imperfections of the clinical data, or because of the confusing character of the lesions, only eleven of these cases could be used with any advantage.

OLDER VIEWS REGARDING THE FUNCTIONS OF THE LENTICULA.

We shall first note briefly the views regarding the functions of the basal ganglia held by some of the older physiological and neurological teachers.

Fifty years ago one of the most relied-upon general works in physiology was that of Carpenter.[1]

An old view which is discussed but antagonized by him was that the thalami in some way were centers for movements of the upper extremity, and the striata for the lower. Carpenter also opposes the idea of Magendie that removal of the striata causes an irresistible tendency to forward progress, whilst the division of the peduncles of the cerebellum occasions the reverse movement.

Discussing the functions of the striatum, Carpenter says that, "According to Longet, Schiff and Lafargue, the results of removal of the corpora striata with the anterior upper part of the cerebral hemispheres, are for the most part negative; for the animal usually remains in a state of perfect stupor, although still retaining the erect position; and it is only when irritated by pinching or pricking that it will execute any rapid movements. No mechanical irritation of the corpora striata produces either sense of pain or muscular movements. No distinct evidences regarding the special functions of either of these ganglionic masses can be gained from pathological phenomena. So far as is yet known extensive disease of either the thalami optici or the corpora striata of one side, produces hemiplegia or paralysis both of sensation and motion on the opposite side. The same result very commonly follows an apoplectic effusion into the substance of either."

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[1] "Principles of Human Physiology," by William G. Carpenter, M.D., F.R.S., F.G.S., Examiner in Physiology and Comparative Anatomy in the University of London, etc., fifth American from the fourth and enlarged London edition, Philadelphia, 1853.

Broadbent,[2] writing in 1876, voiced the views which were held by many about the time and shortly after the time when the study of cerebral localization received its greatest impetus through the suggestions of Hughlings Jackson (1864) and the researches of Hitzig, Fritsch, Ferrier and others (1870-1875).

Discussing the position of the thalamus and striate bodies, with reference to the crusta and tegmentum, after indicating the existence of connections between these ganglia and the other parts of the nervous system, both lower and higher, he expresses the opinion that the striatum is a motor and the thalamus a sensory ganglion for the entire opposite half of the body. With regard to the striatum, like all the earlier writers not separating in his discussion the caudatum and the lenticula, Broadbent holds that this ganglion translates volitions into actions and puts into execution the commands of the intellect. "It selects, so to speak, the motor nerve nuclei in the medulla and cord appropriate for the performance of the desired action, and sends down the impulses which set them in motion. These impulses are transmitted through fibers, and the fibers must start from cell processes in the corpus striatum. A given movement, therefore, must be represented in the corpus striatum by a group or groups of cells giving off downward processes, which become fibers of the motor tract of the cord." When the movement is simple the cell group will be small and the fibers few; when complex the cell group will be large and well defined and the descending fibers numerous.

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[2] Broadbent, Wm. H. British Medical Journal, April 1, 1876.

"Words," says Broadbent, "which require for their utterance the simultaneous co-operation of the muscles of the chest, larynx, tongue, lips, etc., and the exquisite and rapid adjustment of their movements concerned in phonation and articulation, must be represented in the corpus striatum by very large groups of cells, and not in that of one side only but in both."

According to Ferrier[3] it is evident that destruction of the striatum produces a much more complete and enduring paralysis than destruction of the cortical motor centers alone, or of the pyramidal tracts which proceed from them.

He summarizes his views with regard to the corpora striata as follows: "It appears from these facts that the corpora striata proper are centers of innervation of the same movements as are differentiated in the cortical motor centers, but of a lower grade of specialization. The innervation of the limbs in all that relates to their employment of instruments of consciously discriminated acts is dependent on the cortical centers, while for all other purposes involving mere strength or automatism, primary or secondary, the corpora striata with the lower ganglia are sufficient. In man almost every movement has to be elaborately acquired by conscious effort through the agency of the cortical centers, and continues to involve the activity of these centers to a greater or less extent throughout. Hence the destruction of the ganglia of the corpora striata adds little if anything to the completeness of the paralysis which results from destruction of the cortical motor centers alone."

Ross[4] has contributed something to our more exact knowledge of the motor functions of the lenticula in a paper on what is now commonly designated as pseudo-bulbar paralysis, the title of his contribution, however, being labio-glosso-pharyngeal paralysis of cerebral origin. He cites a case from Oulmont[5] with the typical symptoms of pseudo-bulbar paralysis in which the motor and speech phenomena were apparently due to bilateral lesion of the lenticula.

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[3] "The Functions of the Brain," by David Ferrier, M.D., second edition, rewritten and enlarged, New York, 1886.

[4] Ross, James. Brain, July, 1882.

[5] Oulmont. Cited by Lépine, R. Mensuelle de Médecine et de Chirurgie, Vol. 1, 1877.

"An old hemorrhagic focus was found in the right hemisphere occupying the third or external segment, and a smaller focus situated in the second segment of the lenticular nucleus. Similar foci were found in the third and second segments of the lenticular nucleus of the left hemisphere. The internal capsule was not involved in either hemisphere. The medulla oblongata and pons were free from disease."

Ross cites a case of Eisenlohr[6] in which lesions were found in both striata (the lenticular nuclei?) and in the thalami.

Ross also cites the case of Kirchhoff,[7] published in 1881, in which lesions of both the caudatum and lenticula were found, but as the capsule also seems to have been involved in this case, it is not of as much value for our purposes as other cases in which the lenticula alone seems to have been implicated.

One of the most important cases of labio-glosso-pharyngeal paralysis or pseudo-bulbar paralysis from disease of the lenticula which has as yet been described is that reported by Ross himself. In this case the lesions described were as follows:

"The lenticular nucleus of each hemisphere was found post-mortem replaced by a well-defined cystic cavity, containing a clear, straw-colored fluid, the internal capsules being apparently uninjured. Microscopic examination failed to detect any evidence of disease in the nerve nuclei of the medulla oblongata, or descending changes in the pyramidal tracts in any part of their course."

In another case recorded by Ross the lesions present were described as follows:

"The anterior part of the lenticular nucleus of the left hemisphere presented two small cyst-like cavities, containing clear fluid. These cavities occupied the second and third segments of the nucleus, and one of them appeared to encroach to some extent on the knee of the internal capsule. The right hemisphere was preserved in spirit, and was not examined until a fortnight later, when a small cavity was found in the lenticular nucleus of this hemisphere also. This cavity occupied the anterior part of the nucleus, but did not appear to encroach upon the internal capsule." Some descending degeneration, probably due to lesion in the internal capsule, was present in this case.

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[6] Eisenlohr, C. Archiv. für Psychiatrie, Vol. IX, 1878, p. 43.

[7] Kirchhoff. Archiv. für Psychiatrie, Vol. XI., 1881, p. 132.

In a third case recorded by Ross a small cystic cavity was found in the left lenticular nucleus close to the border of the genu of the internal capsule. No secondary degeneration detectable by the naked eye was in the crura. A careful microscopical examination of successive sections of the crura, pons, and medulla oblongata, failed to detect any secondary degeneration of the pyramidal tracts. The bulbar nuclei were healthy. This case, which was complicated with disease of the spinal cord, presented typical pseudo-bulbar paralytic symptoms.

Ross held that his cases did not prove that the lenticula is an independent center for the regulation of all movements of articulation and deglutition. He was also inclined to negative the view that the lenticula is a ganglion of interruption between the cortex and the parts below, and looked with favor on the idea that in the cases of labio-glosso-pharyngeal paralysis recorded by him the symptoms were due to lesions interfering with the paths of conduction from the cortex, although at least two of his three cases seem to have been instances of lesions isolated to the lenticula, these causing disorders of speech and some paresis of the face and upper extremity. His view would appear to be in a general way that of Dejerine that the cases of pseudo-bulbar paralysis attributed to lesions of the lenticula, either unilateral or bilateral, are probably dependent upon undetected lesions of the pyramidal tracts or cortex.

THE VIEWS OF DEJERINE.

In his Anatomy of the Central Nervous System[8] Dejerine in several places goes rather fully into the question of the functions of the lenticula, but his results would seem to be included in the assertion that this ganglion plays only a negative part. He is mostly concerned with denying its connection with the motor and other portions of the cortex by projection fibers, appearing to hold that the lenticula plays no part in the control of movement or in the function of speech. In order that his views with regard to the functions of the striate bodies may be understood, it will be necessary to include a reference to his opinions as to the anterior limb of the internal capsule. Lesions of the anterior seg-

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[8] Dejerine, J. "Anatomie des centres nerveux," Vol. II., fasc. 1, Paris, 1901.

ment of the internal capsule, says Dejerine, are not manifested by any appreciable motor disturbances when they occupy only the anterior half or three-quarters of this segment. These fibers are, he believes, frontothalamic projection fibers. He says that as soon as the knee of the capsule is reached, a distinct symptomatology appears,—that of paralytic phenomena on the opposite side of the body. Secondary degeneration of this part of the capsule is also seen following cortical lesions of the Rolandic zone. Dejerine has shown that the area of degeneration in the anterior portion of the posterior limb of the capsule is correspondingly more posterior, according as the lesion is higher and higher in the Rolandic cortex, his own observations confirming in this respect the physiological observations of Horsley and Beevor on the internal capsule of the monkey.

When the knee and the adjacent portion of the posterior limb of the internal capsule are involved in a lesion, a paralysis of the inferior facial nerve and of the half of the tongue on the opposite side is observed. The fibers arising from the frontal operculum and Rolandic operculum pass through the knee and the adjacent portion of the posterior segment of the capsule. Facial paralysis of capsular origin presents the same features as that of cortical origin, the superior facial is not entirely intact.

When the lesion is bilateral and symmetrical, the clinical picture will be that of pseudo-bulbar palsy. The anatomical localization of the paralysis has been and still is differently interpreted. It is known that this may be of cortical origin (bilateral lesion of the Rolandic operculum), subcortical, capsular, pontile, or bulbar, situated in the course of fibers arising in the motor zone which control the nuclei of the motor cranial nerves. Dejerine does not accept the localization of those who have their views based upon cases in which the lesions were supposed to be limited to the lenticula. In one place he says emphatically that the lenticular nucleus does not send fibers into the foot of the cerebral peduncle and does not receive any from the cortex. Further on in the same work, however, he modifies this statement by saying that the *putamen* does not receive any fibers from the cortex, but that the globus pallidus may do so. The lenticular fibers of projection, he holds, belong to the system of strio-thalamic and subthalamic radiations.

Dejerine states that symmetrical lesions (hemorrhage, softening, either in the putamen or in the lenticular nucleus as a whole) are frequently observed in the necropsies of the aged, who have not presented any symptoms of pseudo-bulbar paralysis during life. He recalls the fact that Oppenheim and Siemerling had shown in 1886 that when they observed a bulbar palsy during life in these cases of symmetrical lesions of the lenticula, there were always also foci of softening in the pons and oblongata. According to Dejerine in all the cases in which there was absence of all pontile or bulbar lesions appreciable by the microscope, the pseudo-bulbar palsy seems to have resulted not from the localization described as lenticular by some; but when these cases are studied by the method of serial microscopical sections, the bilateral and symmetrical lesions of the lenticula are found always to have involved the knee and a portion of the posterior limb of the internal capsule.

He speaks of tracing clearly in the retro-lenticular portion of the internal capsule fibers which traverse the globus pallidus. He says that these may appear to be cortico-lenticular fibers, but suggests that inasmuch as they cannot be followed in the corona radiata, except immediately above the retro-lenticular segment, they may rather be lenticulo-caudate fibers, that is caudate fibers of projection or termination.

Dejerine asserts that in cortical lesions, either extensive or confined to small areas, when the material is treated by the method of Weigert and Pal, the globus pallidus does not appear to undergo any modification, either in the form of atrophy or disappearance of fibers, similar to that which is seen in the thalamus in the same case. He acknowledges, however, that we cannot assert that the globus pallidus possesses in such cases as many radiating fibers, or as dense a network of fibers as in the normal state.

It is further stated by him that in two cases of recent cerebral disease, in one of which the lesion was extensive and in the other limited, examined by the method of Marchi, the globus pallidus and the corpus Luys both received fibers of projection from the cerebral cortex.

In still another place Dejerine states that the striatum, especially the caudate nucleus and the putamen, at least in man, do not receive direct cortico-striate fibers; that they receive only cortical fibers of projection traversing them on their way to the internal capsule (cortico-thalamic, cortico-pontile, cortico-bulbar, cortico-spinal fibers). It seems to be well demonstrated, according to Dejerine, that cortical lesions, whatever may be their situation, their extent, and their duration, do not affect the caudatum and the putamen as they do the thalamus. This contrast existing between the striatum and the thalamus, says Dejerine, was well recognized by Gudden in 1872 as the result of experimental observations. Other well known investigators cited by him have observed atrophy of the striatum following cortical ablation experiments, but this atrophy results probably from the degeneration of fibers of passage and collaterals or injured cortical neurones, and is not comparable with the atrophy, even extreme, in the thalamus under the same circumstances. The very slight diminution in size of the striatum sometimes seen in man following old lesions of early childhood is, in Dejerine's opinion, the analogue of the diminution in volume seen in all these cases in the anterior corresponding half of the encephalon, in the anterior horn of the opposite side of the cord, and in the opposite cerebellar hemisphere. It results as much from degeneration of the collaterals which the system of cortical projection fibers sends to the striatum and the region of the tegmentum, as from the functional inactivity of this region. Dejerine[9] has expressed his views regarding the lenticula also in a recent paper.

Some others, (Schwalbe, Edinger, Sachs, Bechterew, Marinesco). Dejerine remarks, deny the existence of cortico-striate fibers of projection, but depending on the homology of development of the striatum and of the cerebral cortex established by Wernicke, attempt to prove the existence of a cortico-striate fasciculus of association, and as such describe the occipito-frontal fasciculus and collosal fasciculus of Muratoff.

THE VIEWS OF MINGAZZINI.

In the appendix to his monograph, Mingazzini[10] discusses and antagonizes the opinions of Dejerine.

The arguments of Dejerine, says Mingazzini, appear far from decisively proving that the lenticular nucleus does not possess motor fibers, and that therefore a lesion of the same is incapable of producing paralytic disturbance. The argument that the secondary degenerations show that the lenticula does not send fibers into the foot of the cerebral peduncle and does not receive any from the cortex, and its fibers of projection must belong only to the system of the strio-thalamic and sub-thalamic radiations, according to Mingazzini, has little value, since he (Mingazzini) believes that he has shown in the beginning of his monograph that lesions of the lenticula, according to the points at which they occur, seem to give rise to a diverse symptomatology.

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[9] Dejérine J. L'Encephale, No. 5, May, 1907.

[10] Mingazzini, Giovanni. "Sulla sintomatologia delle lesioni del nucleo lenticolare." 1902.

Mingazzini believes that it is not true that in every case in which lesion of the lenticula produces permanent disturbances, there must be existing concealed destruction of the internal capsule. He believes that the arguments of Dejerine might just as well be turned around; in other words, it is just as likely that lesions of the lenticula might be present and escape detection in those cases in which capsular lesions are discovered as the reverse. The arguments of Dejerine that because lesions of the pons and bulb are frequently found in pseudo-bulbar paralysis, therefore these are probably present and the real cause of the paralytic phenomena in cases apparently due to lesions of the lenticula, is easily overcome, according to Mingazzini, if we remember that the symptomatology of cerebro-pontile pseudo-bulbar paralysis is much richer than pseudo-bulbar paralysis of pure cerebral origin; in other words the picture of the two diseases is different.

Mingazzini insists on the point that only certain parts of the lenticula transmit motor and sensory fibers or have motor or sensory functions, and it is therefore easy to understand how lesions may sometimes be found in these ganglia in the old or in others without motor symptoms.

Mingazzini does not accept the opinion of Dejerine with regard to the cases collected from different authors, in which atrophy of the lenticula occurred after lesions or arrests of the cerebral lobes or convolutions, but believes that these cases show that anatomical and physiological connection exists between some part of the cortex and the lenticula. Mingazzini lays particular stress upon the researches of Dejerine, already referred to in our analysis of Dejerine's views, in which he found degenerated fibers in the globus pallidus as the result of cortical lesions, since Mingazzini says it shows that these ganglia receive fibers of projection from the cerebral cortex.

Leaving his criticisms of Dejerine, let us turn now to Mingazzini's positive statements of his own views based upon personal clinico-pathological observations, his pathological findings being both gross and microscopic. In his monograph he first calls attention to the manner in which the question of the function of the lenticula has been largely passed by, or has been altogether slighted by writers on neuropathology and clinical neurology. He refers particularly in this connection to well known authors like Gowers, Oppenheim, von Monakow, and Brissaud. Unlike Dejerine, who attacks the problem of the functions of the lenticula in his great work on the anatomy of the nervous system, with the result of negativing its functional importance, the other authors referred to by Mingazzini have contented themselves with slight or imperfect allusions to the problem of lenticular disease.

Mingazzini, in this monograph, gives details of nine cases of lenticular lesion with necropsies observed by him, and refers to other cases in a preceding publication. We have gone carefully over the notes of these cases, and also the table in which the topography of the lesion and the disorders of articulation and of motility in the face and limbs are summarized. These cases would seem to leave little doubt of the correctness of the conclusions drawn by Mingazzini. One criticism is worth mentioning, however, regarding some of them. Several of these patients were quite advanced in years; some over seventy and others between sixty and seventy. In several of them also abnormal conditions of the meninges and cortex or subcortex were present, as opacities of the membranes, adhesions with decortications, shrinkage of the convolutions, and in one or two instances, actual softening. Making, however, due allowance for these findings and their possible bearing upon the symptoms in limbs, face and speech, we believe that the most important of Mingazzini's conclusions are to be accepted. We shall give these somewhat at length, translating and summarizing his remarks, omitting quotations, as in order to save space we shall not follow his language consecutively and with exactness.

A focus of disease, even of small size, involving only the lenticular nucleus, according to Mingazzini, never fails to manifest itself with motor disturbances. Usually these are dissociated or total paralyses, to which are sometimes added irritative symptoms.

Facial paralysis is very rarely of lenticular origin. Mingazzini has not observed any case of it. He refers to the one recorded by Ross, to which we have already directed attention. Cases of unilateral facio-lingual paralysis and of bilateral facio-lingual paralysis such as is frequently seen in pseudo-bulbar paralysis without involvement of the limbs are also rare. Mingazzini gives three instances of what he regards as facio-lingual paralysis of lenticular origin. In the first the putamen on the left and in the second the putamen on the right in the neighborhood of the genu was softened; in a third a focus of softening was present in the globus pallidus. Mingazzini refers in this connection to the cases of Lépine and Halipre[11] which with others have been criticised by Dejerine.

Mingazzini refers to a form of paralysis intermediate between the pure facial and the brachio-facial paralysis which he had previously reported. The patient presenting this symptom-complex, besides having paralysis of the inferior portion of the right facial, was affected with athetoid movement of both hands. Many lacunar foci were found in the left putamen.

A single example of pure brachio-facial paralysis is recorded. The case was that of a patient affected with paresis of the seventh nerve and of the superior limb of the right side following softening of the anterior portion of the left lenticula.

Facio-crural paresis is very rare indeed. Mingazzini says that he has never seen a pure case of this dissociated paralysis, but refers to a case previously recorded by him in which was present oscillatory tremor of the left superior limb with paresis of the left inferior facial distribution and the left lower extremity. Two foci of loss of substance as large as a millet seed were found; one in the external part of the right putamen and the other in the external member of the globus pallidus. In two cases Mingazzini has observed an irritative paretic disorder of the upper extremity from partial destruction of the lenticular nucleus on the opposite side. In one of these cases the limb affected was spastic; in the other it was the seat of clonic movements. In another case in which softening was found in the middle part of the left putamen, the patient was seized occasionally with clonic movements of the hand and the forearm of the opposite side.

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[11] Lepine, R. Op. cit.

While the forms of hemiplegia, with or without irritative phenomena, are sometimes present in lesions limited to the lenticula, the more frequent syndrome, according to Mingazzini, is motor paresis of the entire half of the body. He records five such cases in the monograph we are analyzing and refers to others previously recorded. The paralytic disturbances often pass without being observed by the patient, says Mingazzini. They apply to the physician complaining of paresthesia, he continues, or of pain along the paretic limbs, or else of vertiginous conditions, or weakness of memory, symptoms evidently due to a diffuse cerebral arterial sclerosis. It is only a minute objective examination, he says, which will reveal certain hemiparesis. While the attack of paralysis may be ushered in by an apoplectic stroke with unconsciousness, the symptoms accompanying this are not so severe and persistent as in ordinary cerebral hemorrhage. The patients are often syphilitic, and probably because of this the attacks are frequently preceded by headache and febrile symptoms. As indicated, much of the paralysis disappears, but a residue can always be found by a careful examination. The paresis or paralysis of the facial is almost always limited to the inferior distribution of the nerve. The superior extremity assumes peculiar attitudes. The forearm tends to bend on the arm, and the hand on the forearm, but these conditions or attitudes are only rarely spastic in character, as is observed in capsular lesions. This point seems an important diagnostic one to the writers who have observed cases presumably of lenticular lesion in which the non-spastic limbs assumed special attitudes, simulating those seen in spastic hemiplegia. The three portions of the upper extremity are usually about equally affected with the impairment of power. Gross oscillatory tremors may result from attempts to use the limb. In the lower limb, as in the upper, the paresis is usually about equally distributed in its three parts. It is rarely, if ever, a complete paralysis. The patient feels as if his paretic leg would give way; sometimes he can walk and sometimes not; when he walks his steps are slow and short. The leg of the paretic side is a little flexed on the thigh, the foot striking the ground, without, however, performing the classic movement peculiar to capsular lesions.

Mingazzini lays stress upon the fact that the majority of his cases with the syndrome of hemiparesis could be referred to softening of the putamen, situated near, and in correspondence with, the anterior limb of the internal capsule and not with the posterior portion. The lesions being somewhat removed from the pyramidal tracts, therefore their effects when these lesions are hemorrhagic cysts cannot be attributed to pressure upon these tracts. In addition, Mingazzini points out that in most of his cases the lesions were foci of softening which did not exercise pressure on neighboring parts. A still further point in favor of the paralytic disorders being due to lenticular lesions and not to pressure exerted upon the capsule is the fact that these affections, even if they are not marked, are persistent and continue until death. Although partial paralyses, they are not transitory.

The motor function of the lenticula, says Mingazzini, deserves to be called supplementary if it is borne in mind that the motor disturbances in comparison to those produced by the destruction of the pyramidal region of the internal capsule are very mild. Mingazzini does not attempt to decide whether the motor fibers which originate in the lenticula join with those of the internal capsule; whether the neurones of which they are part originate in the motor cortex or in the lenticula itself; and whether if they originate in the lenticula they come in contact with neurones derived from the cortex. These are matters for future research to determine, especially by the studies of experimental and other degenerations. His claim is simply that the lenticula is a motor organ of some sort: that it possesses real motor function. He refers to Johansen's faradic excitation of the lenticula as confirming his own results and views.

Disturbances of speech, according to Mingazzini, are often associated with lesions of the lenticula. He says with some reserve that he believes the dysarthric disturbances dependent on lenticular lesions arise only when these lesions are situated on the left side. He has recorded cases in which the dysarthric disturbances were absent even when the lenticula was injured on both sides. His explanation of such observations is that the fibers destined for the movements concerned in speech run in circumscribed zones, as it is true that the fibers destined for the face, the upper and lower limbs, run also in circumscribed zones; in other words, that the motor zone of the lenticula is subdivided as is the cortical motor zone.

Discussing in detail some of his observations regarding speech, Mingazzini says that disturbances of articulation under the form of elisions, initial pricking and bad pronunciation of the dentals were signalized in one case in which the external member of the globus pallidus on the left was destroyed; and in another case in which there was a softening of the left putamen. But it was impossible to find any disorder of speech in still another case in which there was a hemorrhagic focus on the external margin of the right putamen. Disturbances of articulation were found in a case in which there was a loss of substance which occupied the more external limit of the left putamen, on a level with the middle part of the colliculus caudatus; and these were not found in another case in which the hemorrhagic focus occupied the medial and inferior part of the globus pallidus of the left; and not in still another case in which the lesion was in the base of the putamen and in the middle part of the external capsule of the left. These results demonstrate again that the lesions of the right lenticular nucleus have no effect on speech, and that those of the left lenticular nucleus give rise to disturbances of articulation only if situated at fixed points.

VIEWS OF SCHÄFER, VON MONAKOW AND OTHERS.

Coming to one of the most recent physiological works, the views of Schäfer,[12] regarding the functions of the striatum (lenticula and caudatum) are stated in the following manner:

"Cajal describes axones of many of the pyramidal cells of the frontal lobe as passing to the corpus striatum. After destruction of the frontal lobe in dogs and monkeys, Marinesco found numerous degenerated fibers in the corpus striatum and especially in the caudate nucleus. On the other hand, the corpus striatum receives numerous fibers from the thalamus and the sub-thalamic region, and probably sends out centrifugal fibers downwards, along the motor tract; but these have not hitherto been satisfactorily traced; some appear to go to the substantia nigra. The corpus striatum is generally believed to act as a center for the higher reflex movements, and to be in close association with the Rolandic area, but the experimental grounds for this belief are still lacking. Morphologically, the corpus striatum is regarded as a part of the cortex."

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[12] "Text Book of Physiology," edited by E. A. Schäfer, LL.D., F.R.S., Vol. II., Edinburgh and London, 1900.

Von Monakow[13] says that a lesion taking in almost the entire caudate nucleus, leaving the internal capsule free, or even the lenticular nucleus, does not of necessity cause a persistent hemiplegia. He continues that Reichel[14] has shown that a symmetrical softening of both lenticular nuclei may occur without clinical manifestations. As a rule, hemiplegia with the involvement of the facial and hypoglossal occurs in extensive lesions of the caudate and lenticular nuclei. It is seldom complete and only persistent when the internal capsule, that is the premedial portion of it, is implicated. Some areas of softening, either in the lenticula or in the caudatum, usually are latent. Irritating foci, for example tubercles, cause occasionally early hemiplegic contraction. Aside from the hemiplegia,—though together with this in connection with lesion of the caudate and lenticular nuclei—occasionally occur hemianesthesia, tremor, forced laughter, choreiform movements, vasomotor disturbances (elevated temperature, reddening of the skin, etc.) and central pain in the opposite half of the body, especially in the arm. But all these phenomena, as a rapid glance through the literature on focal diseases shows, may occur without direct lesions of the ganglia of the forebrain. Occasionally they appear following lesions in the neighborhood of the posterior internal capsule, in the posterior portions of the thalamus, and in the tegmental region. Nothnagel, for instance, says von Monakow, attributes vasomotor disturbances accompanying hemiplegia to involvement of the posterior internal capsule. Numerous observations speak, however, for the closer relation between elevation of temperature in the paretic extremities of one half of the body and disease of the caudate nucleus in the opposite half brain. Experimental investigations also are favorable to this view. We know that irritation of the caudate nucleus in animals may be accompanied by pronounced, though transitory, elevation of temperature. Various investigators have observed similar elevations of temperature on the opposite side to the foci of disease in man. In such cases edema of the skin was also present in the extremities. In all these cass th lesion, even microscopically, was not limited strictly to the caudatum.

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[13] Von Monakow, C. "Gehirnpathologie," second edition, Wien, 1905.

[14] Reichel. Wiener Medicinischer Presse, 1898. "Diseases of the Caudate Nucleus and Lenticular Nucleus." Cited by von Monakow.

Choreiform movements, says von Monakow, were years ago associated occasionally by this or that investigator with disease processes in the caudate or the lenticular nucleus. Ellischer, Flechsig and others have seen in those who have been affected with chorea and have died, peculiar, strongly refractive, often mulberry-shaped, bodies in the lymph sheaths of the vessels in the portions of the brain mentioned. Wollenberg found similar formations in the lenticula of persons who had not suffered from chorea, and therefore disputes the causal relation between chorea and these bodies. Hebolt observed tremor in the extremities in a case of lesion of the putamen. Such isolated observations, according to von Monakow, are of little value so long as they relate to the chance occurrence of focal disease. At all events, he says, a uniform relation has not yet been established between choreiform movements and disease processes limited to the caudate or lenticular nucleus.

Deroubaix,[15] in a paper on spasmodic laughing and weeping, reports one case with necropsy which is of great interest in connection with the pathology of this emotive syndrome, and with the general discussion of lesions of the lenticula. The patient was a man thirty-eight years old who had had a stroke causing left hemiplegia, but without any aphasia or dysarthria. He exhibited spasmodic laughter followed by weeping and accompanied by associated movements in the paralyzed left arm. Death was from a second stroke. Necropsy showed a large area of softening which had destroyed the lenticular nucleus, the anterior limb of the internal capsule, and all the white matter of the frontal lobe and of the central region, to the cortex on the right side. The thalamus, genu and posterior limb of the internal capsule were not affected.

Some of the conclusions drawn by Deroubaix are as follows: That the fibers concerned with the function of emotive expression probably pass outside of the pyramidal fibers like those concerned with the function of coordination and tonus; that the thalamus is the seat of the automatic movements of emotive expression, especially of laughing and crying; that laughing and weeping are under the control of the cortex; that the cortico-thalamic fibers pass in front of the knee of the internal capsule, probably by the lenticular nucleus. Deroubaix therefore did not attribute the spasmodic laughing and weeping in his case to destruction of centers in the lenticula, but of fibers passing from the cortex through the lenticula to the thalamus. He speaks especially of the facio-thalamic bundle.

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[15] Deroubaix, M. A. Journal de Neurologie, Vol. XI., No. 5, 1906.

With regard to this explanation it is not altogether acceptable, as Mingazzini believes he has shown and as facts supplied by others indicate, that the lenticula is a motor organ and has distributed through it zones and centers of particular kind for movements of various parts of the body, including the movements connected with the expression of emotion. This being the case, destruction of such centers, especially if on both sides, would give rise to such a lack of control of the movements of expression as to cause the involuntary or spasmodic laughing and weeping.

In one or two of our cases of lenticular lesion involuntary emotion was present.

In connection with the question of speech disorder from lenticular lesion it will be observed in the case of Deroubaix that the lesion was right sided.

Pagano,[16] by injecting solutions of curare into the caudate nucleus has concluded that:

(1) Excitation of the anterior third and of the middle third of this nucleus provokes in dogs something very like the emotion of fear. This effect is best brought out when the injections reach the internal half of the organ. All the characteristics of this emotion are present, the gesticulations, the play of the physiognomy, the cardiac and respiratory phenomena, the actions of the intestines, and of the bladder, the state of the pupil, and the effect of threats and noises all go to form conclusive evidence of this emotion.

(2) The excitation of these points, but especially of the middle third, provokes a strong erection of the penis which appears immediately after the injection and persists until death.

(3) The excitation of the anterior extremity of the nucleus

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[16] Pagano. Riv dipatol. nerv. e. Ment., July, 1906. Reviewed by W. W. Ireland in the Journ. of Mental Science, January, 1907, p. 175.

produces an agitation which presents the appearance of fear modified with anger.

(4) The excitation of the posterior third of the nucleus provokes a series of manifestations of anger; the grinning and barking, the readiness to attack and bite, and the whole attitude leave no doubt as to the nature of the emotion.

(5) The excitation of the outer part of the anterior third of the nucleus, besides some emotional disturbance, provokes in a greater degree intestinal and vesical phenomena.

The nucleus caudatus according to these experiments is a center of some of the emotions.

Pagano has shown the existence of distinct motor centers in the cerebellum by the same method.

THE NUMBER AND CHARACTER OF THE CASES STUDIED BY THE WRITERS.

The cases which follow are a few out of a considerable number which have come under the observation of the writers, in which lesion of the lenticular nucleus was found at necropsy. In the majority of these cases the lesion, usually a hemorrhagic cyst or an old area of softening, has not been confined to the lenticula; indeed, in most of them several parts of known functional importance, and others whose functions are still in obscurity, were involved in the destructive area of disease. The reason of this is of course easy to recognize, and resides in the fact that the blood supply to the great basal ganglia is from branches of the middle cerebral artery, which also sends branches to various regions both of the cortex and of the interior of the brain. The writers have realized the same difficulty that has been realized by others in reaching conclusions regarding the functions of the striate bodies, because of the infrequency of lesions isolated to them. Even in the cases chosen to illustrate some of the points of this paper, the lesions present were scarcely in a single one absolutely limited to the lenticula. In two or three, however, this was so nearly the case that they have felt justified in drawing some inferences regarding lenticular function.

The points which the writers have attempted to elucidate or at least on which they have tried to obtain some light through the study of their cases, circle around the subject of the mechanism of cerebral speech and that of the representation of motility.

CASE I. *Apoplectic Attack Causing Right Hemiplegia and Loss of Speech—Partial Recovery of Motility and Speech—Some Mental Slowness and Confusion—Dysarthria—Partial Word Dumbness—Word Deafness Present but only Slightly Marked—Hemorrhagic Cyst involving the entire Lenticula, the white matter of the Insula and the anterior part of the First Temporal Convolution, and slightly the Posterior Limb of the Internal Capsule.*

M. S., aged forty-eight years, white, born in Ireland, was admitted to the men's nervous wards of the Philadelphia General Hospital August 21, 1903.

On admission the patient complained of headache and also of more or less weakness and helplessness in his right leg and at times in his right arm.

No family history and no history of important illness previous to that producing his condition on admission could be obtained.

The patient made a statement that he had had fits after an injury to his head, after which he had also developed some difficulty of speech.

He also said that three months ago he came home feeling in his usual health, and after supper became dizzy and fell on his face. He said that he did not know anything for two weeks, but when he recovered his mind, he found that he could not use his right leg or arm and could not speak at all. He remained unable to speak for about two months, since which time his power of speech had gradually returned, but he still spoke with difficulty.

The patient was a hearty, robust-looking man. His tongue was moist, slightly coated; pulse of good quality. His heart action was irregular. The lungs showed good expansion.

The pupils were equal, reacted well to light, but slowly to accommodation.

Examination made September 16, 1903, showed but little loss of power in the right arm or leg; the grip on the right side was, however, slightly impaired. Station was good and gait steady. The reflexes were normal. No note as to sensation was recorded.

On February 24, 1904, a note was made by one of the physicians in attendance which stated that in his last apoplectiform attack he had some spasm in the right arm and leg; and also that he had sensory aphasia; and although he did not miss any words he occasionally missed a syllable.

On February 25, 1904, the record stated that his gait was fairly good, but slightly stiff; that he dragged the right foot slightly. The knee-jerks were spoken of as "spastic" with slight attempts at clonus on both sides; station was good with the eyes open or closed; the pupils responded to light and accommodation; no palsies of ocular muscles were present; the tongue protruded to the right; the naso-labial fold was slightly deeper on the right side. The grip was now said to be better on the right than on the left side.

The patient's mentality was noted as "slow"; he required more time to comprehend questions. He could not read nor write because of lack of education. He appreciated the purposes and uses of objects. He recognized a book and named it as "book." He called a gold watch a "dollar." When pointed to the time, which was 12.20, he said it was "after two"; he could not specify the minutes. He could say the days of the week slowly. When told to close his eyes he grasped his hand; when asked to point to a chair, he picked up his hat. He easily confused the name of one object with that of another. He recognized what spectacles were and said that he needed them himself; he could not, however, name them. He could not name a lead pencil. He recognized a pen, could not name it at first but after a little effort was able to name it. He was also asked to name various other objects, as a key, a ring, and gloves, but failed to do so; but he named some objects of wearing apparel correctly.

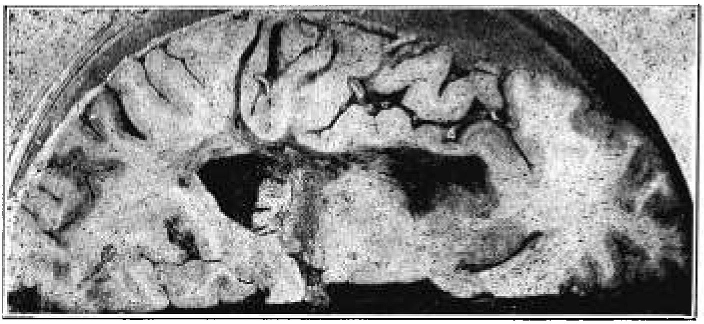

Fig. I. Cyst of the Left Lenticular Nucleus and External Capsule. Motor Aphasia Incomplete (Case I).

Astereognosis was not present.

On December 21, 1905, he was found in a state of collapse and the following facts were noted: His eyes were turned upwards and oscillated slightly from right to left; the face was drawn slightly towards the left side. The right arm and leg dropped lifeless when raised. There was some spasticity in the right arm. The reflexes were exaggerated. On December 22, 1905, the patient was conscious again and seemed to understand all that was said to him and a note said "were it not for his partial aphasia would be able to answer intelligently."

The heart sounds were very good, the second sound being exaggerated, especially the aortic. Pulse at the wrist was strong, of rather high tension and moderately rapid. The patient did not respond to pin pricks. Upon catheterization only one half ounce of urine was obtained.

On December 23, 1905, the patient was in fairly good condition at 10 A.M. At 11.15 he was found rigid, with legs extended and arms held close to the body, the forearm being flexed on the arm. His face was markedly cyanosed. The jaw was rigid, respirations had ceased and the pulse could not be felt at the wrist. The heart sounds could not be heard. A minute or so later the patient relaxed and was dead.

A section made at the level of the upper part of the basal ganglia revealed a cyst in the left hemisphere, which had implicated the middle and posterior part of the left lenticular nucleus, and extended to, but did not involve, the cortex of the island of Reil. It extended ventrally to 0.5 cm. (1-5 in.) from the anterior limit of the lenticula. It extended to, or possibly slightly involved, the posterior limb of the internal capsule. At a level 0.5 cm. (1-5 in.) lower the cyst extended from its posterior end into the anterior part of the white matter of the first temporal convolution, but did not involve the cortex, nor the posterior half of the first temporal convolution.

The temporary right hemiplegia with residual very moderate hemiparesis was probably due to the lenticular lesion, although it is possible it may have depended on the slight involvement of the internal capsule. The temporary, somewhat complete, aphasia, was probably due to the effects of this lesion on neighboring parts, some of the motor defect in speech being, however, probably due to the lenticular lesion. The partial word dumbness could not have been due to lesion of Wernicke's zone, as only the anterior part of the first temporal convolution was involved. As the posterior part of this convolution is considerably higher than the anterior part, the fibers from the former could not have been greatly involved by the extension of the cyst at a low level into the anterior part of the first temporal convolution. It is more probable that some of the symptoms are to be attributed to injury of fibers in the external and extreme capsules which stand in relation to the posterior part of the first and second temporal convolutions.

CASE 2. *Apoplectic Attack causing Right Hemiplegia and alost complete aphasia—Only a few Recurring Utterances retained—Marked Word Deafness at First—Probably some Word Blindness which soon Disappeared—Power of Humming Melodies retained—Continuous Improvement under Systematic Training—Spontaneous Language largely regained—Ability to write with unparalyzed arm acquired—Mentality practically unaffected—Death after four years from Thrombosis of Superior Mesenteric Artery—Dura thickened and adherent—*

Cyst destructively involving the Insula, the Lenticula, the Anterior and Posterior Limbs of the Internal Capsule, the External part of the Thalamus, and the White Matter of the Anterior Half of the First Temporal Convolution at a little distance from the Cortex—The Area of Broca and Subjacent White Matter and Wernicke's Zone not involved.

This case is one, the clinical details of which were in part given by one of us, Dr. Mills,[17] in a paper on the treatment of aphasia by training, published nearly two years before the death of the patient. A contribution giving some facts regarding his condition and attempts made at training him for his aphasia before he was seen by Dr. Mills has also been published by Mrs. Kate Herman.[18]

In the account which follows the writers have summarized the most important facts bearing upon his aphasia and hemiplegia from the papers just referred to, and also from communications received from the patient's former secretary, Miss Dorothy C. Falk, and his former professional and business associate, Dr. H. C. Hays. As this contribution is intended to deal more especially with the functions of the lenticula and the symptomatology resulting from a lesion of this ganglion, the writers have thought it better to concentrate attention upon the facts demonstrating his type of aphasia, rather than upon the efforts at re-education of his language and the results of these efforts. The case is unusually valuable because of the fulness of the history obtained from the different sources to which reference is made, and also because of the intelligence and education of the patient which allowed a more complete record of his speech defects.

The patient was a physician, forty-five years old, residing in one of the western states. In July, 1902, he had an attack of right hemiplegia with almost complete aphasia. He was first under the instruction of Mrs. Kate Herman from June 15, 1903, to July 15, 1903. At the beginning of this period his vocabulary consisted of only four expressions, namely "yes," "no," "but," and "O God." "Yes" and "no" were commonly used correctly. He sometimes added the word "but" to "yes" or "no" apparently when the "yes" or "no" did not seem to him to express his meaning, or in order to give some force to what he was trying to say. At other times he would use the expression "O God." He often used pantomime in an unmistakable manner. When he got possession of a few useful words and phrases he discontinued this expletive. Dr. Mills does not recall his having used it when he was under his care. His memory for persons, places and occurrences was good. He was often very insistent in his efforts to make himself understood. His instructor was soon convinced that his periods of concentration should be short and his exercises for improving his language varied. Word deafness was quite marked at this time, but was not complete. He seemed at times to be partially word blind, but this defect was probably comparatively slight if it really existed, as even early in the efforts to train him he was much helped by seeing the words which were used in training him either in script or printing. It was noted that he could assist himself in understanding what was read from a newspaper by tracing the words with a pencil as the paper was being read.

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[17] Mills, Chas. K. "The Treatment of Aphasia by Training." Journ. of the Amer. Med. Association, Dec. 24, 1904.

[18] Herman, Mrs. Kate S. "Heilung der Aphasie durch den Artikulationsunterricht." "Eos." Vierteljahrschrift für die Erkenntniss und Behandlung jugendlicher Abnormer, Heft 3, 1906.

When he heard music which he knew, he would hum the melody and occasionally articulate some of the words of the song. One day when he was trying to relate an historical occurrence he could not pronounce the word "Maryland" intelligibly until it fortunately occurred to him to murmur the song, "Maryland, my Maryland."

His voice was not impaired,—a rather interesting fact in connection with the functions of the lenticula. He was trained systematically after the manner used with dumb children. The formations and combinations which he found most difficult were those with which such children struggle most, namely, *k, g,* and *ng; th* also gave trouble. He was made to repeat the names of objects in his environment, and his teacher states that the patient would succeed one day in using such words as *house, cow, tree, bird,* etc. and at another time would be unable to recall these, unless helped a little at the beginning. Gradually verbs were taken up and longer and longer sentences tried. With his first instructor, as later with Dr. Mills, it was found that he experienced his greatest difficulty with adjectives, pronouns and articles, and words generally which expressed no mental image.

He was taught to write with his left hand, making continuous and notable progress. He repeated aloud what his instructor wrote for him.

When he first consulted Dr. Mills in October, 1903, his right-sided paralysis, though still marked, was much improved. No loss of sensation and no affection of the bladder or bowels were present. The face was the seat of a moderate right-sided paresis, the tongue not deviating to either side. The deep and superficial reflexes were of the usual type found in cerebral hemiplegia —exaggerated knee-jerk with front tap and ankle clonus and the Babinski response being present on the right side.

When tested with Wyllie's physiologic alphabet, it was found that he could repeat the vowels *a, e, i, o* and *u* well, having been previously trained in their pronunciation, but he could not remember them from day to day. In like manner he could repeat the consonants of the alphabet, but could not recall them spontaneously. He was partially word deaf, having regained word hearing to a considerable extent by time and training. He could understand most familiar expressions, but if a strange or unusual word were used he did not understand it. He was also apparently partially word blind, having relearned to read considerably before coming under observation. With his left hand he could write the words that he was able to read. He could not understand the meaning of such prepositions as *of, to,* and *for,* if pronounced or read. He could not spontaneously write these words when asked, but could copy them.

In connection with a study of his reacquisition of language, particular attention was paid to the degree in which he regained different parts of speech. It was noticeable that in his efforts to talk he chiefly used his nouns and verbs, occasionally employing a pronoun. The words which expressed the qualities of things, which join and relate things to each other and to actions,—in other words, adjectives, adverbs, conjunctions and prepositions,—were still almost entirely eliminated from his vocabulary. Auxiliary verbs like *to have,* and *to be,* in all their modes and tenses, were apparently without any meaning to him. The word *is* or *was* occurring in a sentence seemed to worry him. In brief, this patient was a hemiplegic aphasic, the motor aphasia having at first been almost complete; the sensory aphasia partial. An appreciable degree of recovery from his aphasia had occurred from the first under the method of training by repetition and by efforts at spelling, reading and writing.

Persistent efforts at training were made. The measures employed were the facial and other gymnastic methods used with the deaf and dumb, the method of repetition from dictation, of copying, the use of Wyllie's physiologic alphabet, of a phonetic reader, a language primer and grammar. An interesting point in the history of the attempts at this man's re-education in speech was the fact that he made much use of a small dictionary. He made considerable advance by looking up the definitions of words, and when he found words and expressions in the definition which he could not comprehend, these in their turn were looked up in the appropriate places.

After the patient's return home communications were received from time to time regarding him. He steadily improved, although at first his progress was comparatively slow. In a short time he was reading his newspapers and magazines and understanding fully what was in them. Six months after he left Dr. Mills word was received that he was acquiring more voluntary expressions every week. A noticeable feature now was that when he was told to say anything instead of repeating it verbatim, he would express the idea in his own words. He read much and played whist and other games, and had just finished transacting some business for the institution, the first which he had done of any importance since his illness. A little later, in describing his improvement, the physician associated with him in his work, said that his use of words to express an idea was much more general than had been the case heretofore. He now expressed in his own words answers to questions in the form of a sentence rather than a single word, and it was noticeable that he was able to use a different word to express the same idea. At times he made sentences of considerable length, using nouns, verbs, adjectives, prepositions, etc. Thorough and systematic training with the assistance of his instructors and members of his family was kept up, the patient helping the work very much by his own determination and originality in methods. He continued to use his dictionary. His teaching was much along the line of both what is known as the word method and also that of spelling by using each letter as in the older method.

Many of the details of the methods used to improve his language, and of the time spent in these efforts will be better reserved for an additional paper. With regard to the re-education in language this case is the most interesting that has ever come under our observation. Nearly four years elapsed between the time of the apoplectic attack causing his hemiplegia and aphasia, and his death. During much of that time with interruptions sometimes extending over a few weeks, or even months, the earnest, formal retraining of his speech was carried on, and even when this systematic instruction was not resorted to, the patient, a man of much originality and energy, was always trying to re-educate himself in the use of language. His former secretary calculated that during the four years he was given about one and one half years of training. As a rule only one to two hours daily were given to formal instruction.

It is interesting to note in connection with the question of intellectual deficit in aphasics, which has become so prominent through the views of Marie, that this patient retained throughout his mental vigor and general capacity. His deficiencies seemed to be entirely those conditioned by his defects of speech, meaning by this, of course, his word deafness, his early slight or moderate word blindness, and his difficulty in correlating concrete concepts with each other as well as his very pronounced motor aphasia. About nine months after his attack the sanitarium, of which he was the proprietor and chief physician, burned down. Measures were at once taken to rebuild it, and as bearing on his mental condition, his secretary wrote Dr. Mills that he was always able to figure on any proposition just the same as before his illness but he could not speak it, all his figuring for his sanitarium being done in a very accurate and clearsighted manner.

The improvement in his speech was continuous up to the time of his last illness.

He had no second apoplectic attack. He had, however, three distinct epileptiform seizures, at long intervals, during the last two or three years before his death. No increase either of his paralysis or of his aphasia occurred as the result of these attacks.

His general mental state was good. At times there were indications of moroseness, some irritability of temper and impatience when he was not immediately understood, and occasionally when much discouraged, he spoke of suicide. He showed no marked change in disposition which could be noted as altogether different from his former self. His general physical health remained good.

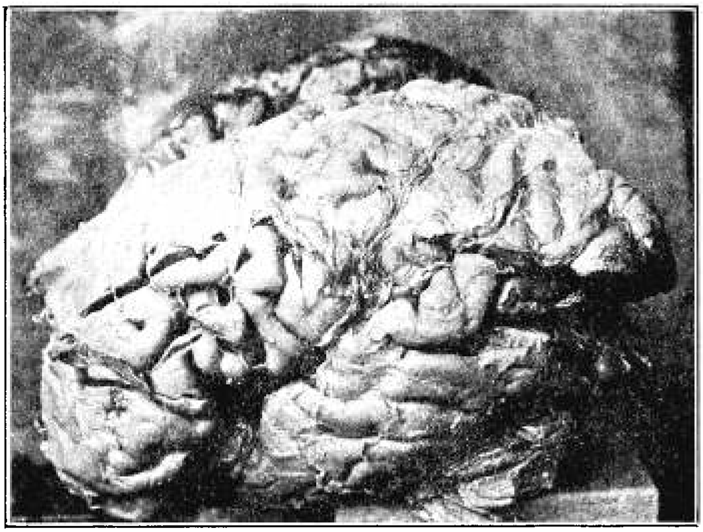

Fig. 2. A case of marked motor and sensory aphasia. The area of Broca is intact and the subcortical white matter was not involved in the primary lesion (Case 2).

The conditions and symptoms preceding his death could in no way be attributed to the cerebral lesion causing his aphasia and hemiplegia, although the changes found in the aorta and the superior mesenteric artery might have some relation to the alterations in the vessels of the brain which were present at the time of the cerebral attack. He died about four years after the onset of his aphasia and hemiplegia, from thrombosis of the superior mesenteric artery, as determined at the post-mortem examination. His symptoms were those of shock and great abdominal pain.

The necropsy was made by Dr. Philip Hillkowitz of Denver, Colorado, at which place the patient was at the time of his death. We are much indebted to Dr. Hillkowitz for the account of the gross findings. The body was well nourished, with considerable adipose tissue; muscular development good; the heart showed slight thickening of the mitral valve; numerous small plaques were found on the aorta, above the valves and around the opening of the coronary arteries. Marked hyperemia was present around the opening of the right coronary artery. The lower lobe of the left lung was strongly adherent to the chest wall and diaphragm. The left kidney was very much puckered on its surface. A slight scar was found on the external border, about 1 cm.

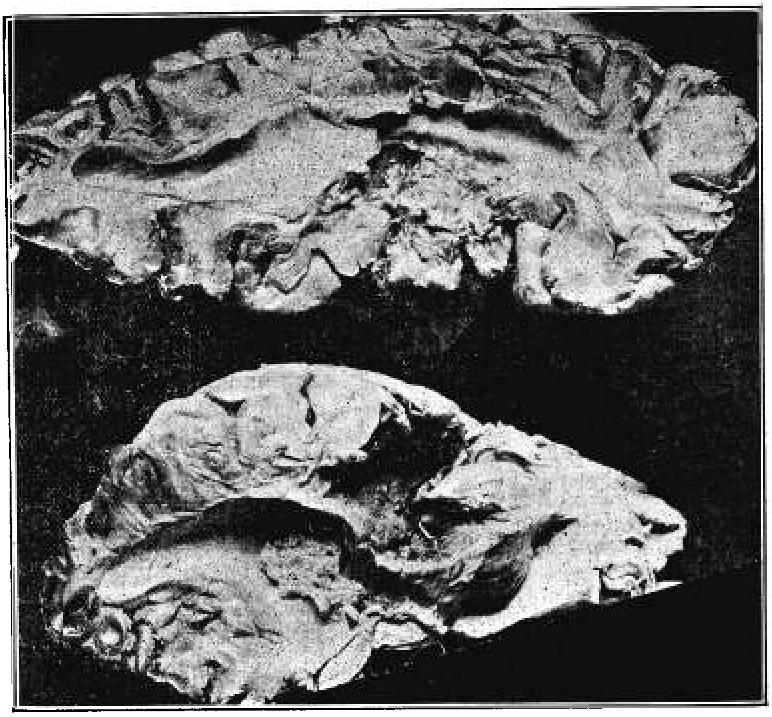

Fig. 3. The lenticular nucleus and the island of Reil were destroyed. The sclerosis extended anteriorly to the white matter of Broca's area, and posteriorly to the posterior extremity of the island of Reil (Case 2).

(2-5 of an in.) in diameter, extending into the interior. The cortex was slightly diminished and was pale in color; the right kidney was similar to the left with the exception of the scar. The intestines showed a dark purple color extending all over both the large and small intestines. The superior mesenteric artery showed a thrombosis which could be followed in the branches of the vessel.

The dura was much thickened and adherent in many places to the membranes beneath. The membranes over the occipital lobe were largely congested. An elliptical cavity 5 x 2.5 cm. (2 x 1 in.) was found in the left cerebral hemisphere. The description of the parts destroyed by this lesion is given in the following paragraph after careful study of the specimen by Dr. Spiller, the brain having been forwarded, after hardening in formalin, to the Laboratory of Neuropathology of the University of Pennsylvania by Dr. Hillkowitz, who however had made an examination at the time of the necropsy. The description of the position and extent of the lesion differs in only one particular from that furnished by Dr. Hillkowitz, although this is one of some importance. Dr. Hillkowitz speaks of the lesion as involving a portion of the inferior frontal convolution (presumably meaning by this the third frontal). Although the cyst extended close to the third frontal it did not involve the cortex of this convolution.

A large cyst was found in the left cerebral hemisphere. In a horizontal section through the lateral ventricle and upper part of the basal ganglia, the sclerosis was found to extend to 1.5 cm. (3-5 in.) from the cortex of the convolution posterior to the ascending limb of the fissure of Sylvius. The area of Broca and subjacent white matter were intact, although fibers passing to or from the foot of this area were probably implicated. The cyst had destroyed the island of Reil, the whole of the lenticula, the anterior and posterior limbs of the internal capsule and the external part of the thalamus. The head of the caudate nucleus was very slightly affected. The gray matter of the first temporal convolution was not involved, although the white matter was involved at a little distance from the cortex.

The part of the first temporal convolution destroyed was the white matter of its anterior half. To be exact, the posterior border extended to the beginning of the innermost part of the middle portion of the first temporal convolution, in a section made through the upper part of the basal ganglia.

This case, although one of great value in connection with the discussion of the subject of aphasia, loses some of its importance as regards the functions and symptomatology of the lenticula, because of the fact that not only was this ganglion destroyed, but so many other functionally important regions. Any speech or paralytic disorder if dependent upon the lenticular lesion, was probably covered by the effects of destruction of other parts. Broca's convolution was not involved, as was not the Rolandic operculum.

THE LENTICULAR ZONE OF MARIE AND MOTOR APHASIA.

Some light is thrown on several of the points of great interest in connection with the current discussion of the subject of aphasia, springing out of the somewhat revolutionary contributions of Marie, by the clinical and pathological facts observed in the case just described. Marie has laid much stress upon the lenticular zone. In defending himself against those who have charged him with giving too much importance to the lenticula in his theories of the pathogenesis of motor aphasia, he insists that the lesions producing the train of symptoms to which he has called attention are not due to isolated lesions of the lenticula, but to it and to those portions of its environment which he includes in this lenticular zone. It is important, therefore, to know just how he defines this zone.

In a horizontal section of the brain, he says, a transverse line drawn from the anterior angle of the insula to a corresponding point of the lateral ventricle will give its anterior border, while a line from the posterior angle of the insula to a corresponding point of the lateral ventricle will give its posterior limit. Thus he includes within this zone more than half of the thalamus, both limbs of the internal capsule, the greater part of the caudate nucleus (all except a part of the tail), the lenticular nucleus, and the island of Reil with the subjacent white matter. Marie does not attempt to distinguish between the symptoms of lesions of the striatum and of the white matter and cortex of the island of Reil. He defines his position concisely as follows:

A lesion implicating only the zone of Wernicke causes the aphasia of Wernicke;

A lesion of the lenticular zone causes anarthria, which is merely the pure motor aphasia of the writers, believed by them to result from a lesion of the white matter beneath Broca's area with the cortex intact. Articulate speech alone is affected, internal speech is preserved; the patient reads and writes and understands without difficulty spoken speech.

A lesion implicating the zone of Wernicke together with the lenticular zone causes the aphasia of Broca (great impairment or loss of spontaneous speech, of reading and writing, and more or less impairment of the comprehension of spoken language).

The case just given fulfills these requirements for the aphasia of Broca. The patient was motor aphasic and agraphic in large measure and had great impairment of the power of reading, According to widely accepted opinion, the case should have exhibited also pure word dumbness and pure word deafness, because the lesion did not extend to the cortex either in Broca's area or Wernicke's zone.[19]

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[19] Marie, Pierre. Revue de Philosophie, 1907.

(To be continued.)